

ary incontinence. Patients may not have headache and may have no apparent signs of increased intracranial pressure. There may be preceding events such as subarachnoid hemorrhage, trauma or meningitis. In some cases the condition may develop coincident with the growth of a neoplasm; in other instances there is no recognizable cause.

Data derived from pneumoencephalographic and isotope cisternographic studies may suggest that normal-pressure hydrocephalus represents a type of communicating hydrocephalus with an insufficiency of the subarachnoid spaces over the convexities, resulting in an impairment in the circulation and absorption of spinal fluid. Computed axial tomography may show large ventricles often accompanied by obliteration of the subarachnoid cisterns and absence of evidence of cortical atrophy. Isotope cisternography frequently elucidates the CSF pathways in hydrocephalus. The isotope after injection into the lumbar sac will reflux into the ventricular system in patients with communicating hydrocephalus, while in normal people it will continue above the hemispheric convexities. False-negative and false-positive results are common, however, and the presence of ventricular reflux has not been clearly shown to determine which patients are likely to improve after a shunt. Water-soluble contrast agents such as metrizamide used in conjunction with computed tomography may provide a reliable image of the CSF pathways in patients with hydrocephalus.

Surgical therapy has been the most efficacious treatment, with ventriculoperitoneal shunt being so far the most widely used of many different procedures that have been advocated for the amelioration of hydrocephalus.

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## CSF Leak After Head Injury

CEREBROSPINAL FLUID (CSF) leak, and the attendant risk of central nervous system infection, is a potentially devastating complication of simple basilar skull fractures. CSF leak can be difficult to recognize soon after injury. Because CSF draining from the nose or ears is admixed with blood, which contaminates the results of dip-stick glucose testing or chemical analysis, laboratory tests are usually

indeterminate. Although the halo sign (a drop of fluid on filter paper forming a clear or pink ring of CSF around a discrete, red circle of blood) may provide early evidence, CSF leakage becomes more obvious 12 to 18 hours after injury, after overt bleeding ceases.

Conservative measures can slow or stop CSF loss. Elevating the head and avoiding Valsalva maneuvers, bending or lifting diminish CSF hydrostatic forces. Fluid restriction (20 ml per kg of body weight a day for adults) and acetazolamide (250 mg daily) diminish CSF production. We administer 16 million units of penicillin daily in adults (ampicillin in children) while leakage is active, though this treatment has not been proved efficacious and is not used in all institutions.

If leakage persists longer than four days, we drain CSF for three to five days, using a fine lumbar subarachnoid catheter introduced percutaneously via a Tuoy needle found in spinal epidural anesthesia sets. This temporary CSF diversion usually stops the leak, but it often causes severe headache and requires caution. If leakage continues longer than 14 days, the exact site of leakage must be found and surgical repair considered. X-ray studies are of minimal value. Thin-section computerized tomography (CT) or complex polytomography may define the fistula. Isotope cisternography sometimes locates a lesion to a general area, but metrizamide isotope cisternography with a high-resolution CT scanner will show the exact site of leakage in most cases.

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## New Surgical Approaches to the Numb Face

COMMON CAUSES of facial numbness include exposure to cold, hyperventilation, hysteria, tumor, stroke, demyelination and trauma. Some are reversible, but others are not and treatment may vary from no treatment to complicated intracranial surgical procedures. Accurate diagnosis can usually be made by examination in the office.

Bilateral numbness of the face without other sensory or motor deficits is commonly caused by hyperventilation or hysteria. While unilateral numbness may be caused by hysteria, it may also be associated with a specific lesion of the trigeminal nerve, ganglion or brain-stem nuclei. If numbness corresponds anatomically to one or more of the